

The Hematology Playbook: A Protocol-Driven Guide to Treatment

Evidence-based management of anaemias, malignancies, and disorders of hemostasis.



A Standardized Approach to Hematological Disease Management

This guide is structured into four core modules, each providing standardized treatment protocols for major categories of blood disorders. Every condition is detailed with first-line therapy, second-line options, and critical, high-yield clinical notes.



1. Anaemias

From common deficiencies to complex hemoglobinopathies and hemolytic states.



2. Haematological Malignancies

A systematic review of treatment strategies for leukaemias, lymphomas, and paraproteinemias.



3. Myeloproliferative & Myelodysplastic Disorders

Management of clonal hematopoietic conditions, from polycythemia to marrow failure.



4. Disorders of Hemostasis

Protocols for managing both bleeding and thrombotic disorders, including coagulopathies and hypercoagulable states.

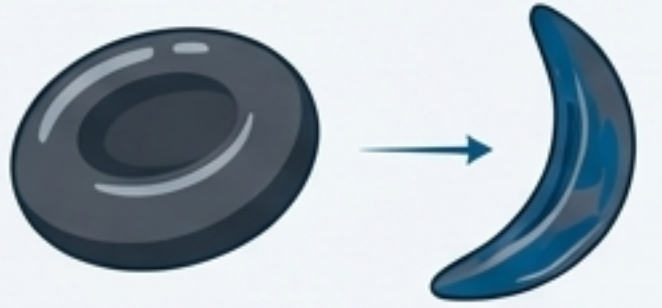
Protocols for Deficiency & Inflammatory Anaemias

Disorder/Condition	1st Line Treatment	2nd Line & Other Considerations	High-Yield Notes & Protocols
Iron Deficiency Anaemia	Oral Iron Replacement: Ferrous sulphate 200 mg 3x daily for 3-6 months to replete stores.	Parenteral Iron: For malabsorption, chronic gut disease, or intolerance to oral iron (ferric isomaltose or ferric carboxymaltose preferred). Transfusion is reserved for severe symptoms (angina, heart failure).	Diagnosis: Serum ferritin is the best single test. Investigation: In men and post-menopausal women, investigate upper and lower GI tract for blood loss. Expected Response: Hb should rise by ~10 g/L every 7-10 days.
Anaemia of Chronic Disease (ACD/AI)	Treat the underlying disorder: Management of the chronic infection, inflammation, or neoplasia is primary.	High-dose IV Iron: Trials are underway to bypass hepcidin-induced blockade. Erythropoiesis-Stimulating Agents (ESAs): May be considered.	Pathophysiology: Driven by hepcidin, which traps iron inside enterocytes and macrophages. Diagnosis: Serum iron is low, but ferritin is normal or high. Can be distinguished from iron deficiency by soluble transferrin receptor levels (normal in ACD).
Megaloblastic Anaemia (B12 Deficiency)	Hydroxycobalamin 1000 µg IM: 6 doses 2-3 days apart, then 1000 µg every 3 months for life (for malabsorption).	Oral B12 Replacement: Sufficient for dietary deficiency. Transfusion: Use with extreme caution due to risk of cardiac decompensation. If needed, give 1 unit slowly with diuretic cover.	Neurological Disease: Associated with B12 deficiency (but not folate). If present, give 1000 µg IM on alternate days until no further improvement. Monitor: Check for fall in potassium and depletion of iron stores upon starting treatment.
Megaloblastic Anaemia (Folate Deficiency)	Oral Folic Acid: 5 mg daily for 3 weeks for acute deficiency.	Maintenance/Prophylaxis: 5 mg once weekly for maintenance. Prophylactic folic acid is critical in pregnancy (prevents neural tube defects) and in chronic hemolytic anaemias.	Initial Treatment for Severe Megaloblastosis: If cause is unknown, <i>always</i> treat with both B12 and folic acid to avoid worsening neurological features of B12 deficiency. Diagnosis: Red cell folate is a more accurate indicator of stores than serum folate.

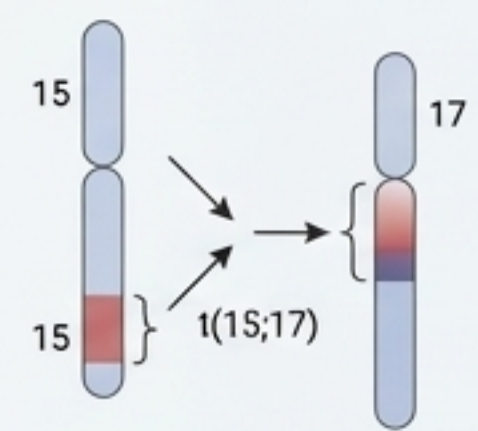
Management of Haemolytic Anaemias

Disorder/Condition	1st Line Treatment	2nd Line & Other Considerations	High-Yield Notes & Protocols
Hereditary Spherocytosis	Folic Acid Prophylaxis: 5 mg daily for life.	Splenectomy: Consider in severe cases with complications (anaemia, gallstones). Delays until after age 6 if possible.	Crises: Aplastic (parvovirus B19), Megaloblastic (folate deficiency), Haemolytic (infection). Diagnosis: Blood film shows spherocytes; Direct Coombs test is negative. Osmotic fragility test or eosin-5-maleimide binding tests confirm.
G6PD Deficiency	Avoidance of Precipitants: Stop any offending drugs (e.g., dapsone, sulphonamides, primaquine) or foods (fava beans). Treat underlying infections.	Acute Transfusion Support: May be life-saving during a severe haemolytic episode.	High-Yield Feature: X-linked disorder. Characterized by episodic, drug- or infection-induced intravascular haemolysis. Blood Film: Shows "bite cells" and Heinz bodies (with supravital stain).
Warm Autoimmune Haemolytic Anaemia (AIHA)	Prednisolone 1 mg/kg orally. Response may take up to 3 weeks. Taper slowly once Hb normalizes.	Second-line: Rituximab. Third-line: Immunosuppressants (azathioprine, ciclosporin), or splenectomy.	Diagnosis: Confirmed by a positive Direct Coombs Test (detects IgG/complement on RBCs). Spherocytes on blood film Transfusion: Use least incompatible blood if life-threatening; risk of reactions.
Cold Agglutinin Disease	Supportive Care: Keep extremities warm. Folic acid supplementation. Treat any underlying lymphoma.	Rituximab (+/- Fludarabine if clonal abnormality is detected). Transfusion: Blood must be administered via a blood-warmer.	Pathophysiology: IgM antibodies cause agglutination at low temperatures. Associated Conditions: Can be secondary to <i>Mycoplasma</i> or EBV infection, or a low-grade B-cell lymphoma. Lab Artefact: May cause a spuriously high MCV.
Paroxysmal Nocturnal Hemoglobinuria (PNH)	Supportive Care: Transfusion, folate supplements, anticoagulation for thrombosis.	Anti-complement Monoclonal Antibodies: Eculizumab or ravulizumab are now standard care; they reduce haemolysis, transfusion needs, and thrombotic risk.	Key Features: Rare acquired clonal disorder causing intravascular haemolysis, thrombosis in unusual sites (hepatic/abdominal veins), and bone marrow failure. Vaccination: Patients on C5 inhibitors <u>must</u> be vaccinated against <i>Neisseria meningitidis</i> .

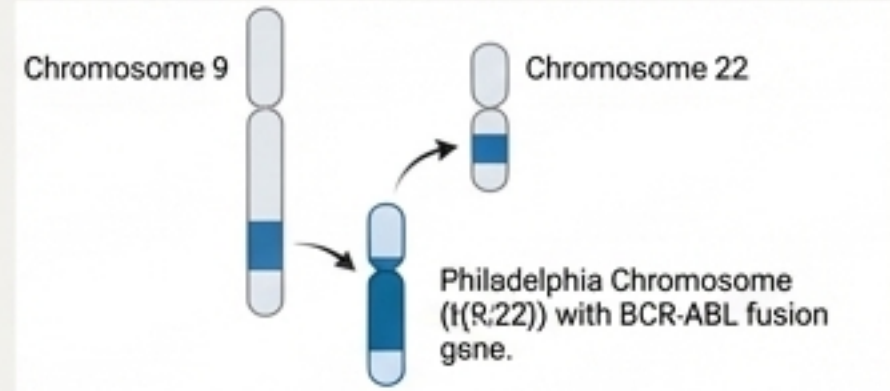
Protocols for Hemoglobinopathies

Disorder/Condition	Management of Acute Crises	Long-Term & Prophylactic Management	High-Yield Notes & Protocols
Sickle-Cell Anaemia (HbSS)  Normal RBC Sickled Cell	Vaso-occlusive Crisis: Aggressive rehydration, oxygen, adequate analgesia (often opiates), and antibiotics. Sickle Chest Syndrome / Stroke: Exchange transfusion may be life-saving. Aplastic Crisis (Parvovirus): Simple top-up transfusion.	Prophylaxis: Daily folic acid, penicillin prophylaxis, and routine vaccinations (hyposplenic state management). Hydroxycarbamide: Increases HbF, reduces frequency of severe crises in children and adults. Crizanlizumab: P-selectin inhibitor to reduce vaso-occlusive crisis. Allogeneic HSCT: Potentially curative.	Pathophysiology: Hypoxia, acidosis, dehydration, or infection precipitates sickling, leading to vaso-occlusion and haemolysis. Screening: Transcranial Doppler ultrasound can identify children at high risk for stroke.
Beta-Thalassaemia Major	Transfusion: Regular transfusions are required to maintain Hb >100 g/L and suppress ineffective erythropoiesis.	Iron Chelation Therapy: Essential to prevent fatal iron overload from repeated transfusions. Folic Acid: 5 mg daily supplementation. Splenectomy: May be required for mechanical problems or excessive transfusion needs. Allogeneic HSCT: Curative option for selected children.	Diagnosis: Profound hypochromic, microcytic anaemia after 4-6 months of age. Hb electrophoresis shows absent/reduced HbA and raised HbF. Prevention: Antenatal screening and diagnosis via chorionic villus sampling.
Alpha-Thalassaemia (HbH Disease)	Transfusion: May be required during periods of increased haemolysis or stress.	Folic Acid Supplementation. Avoid Iron Therapy unless deficiency is proven.	Genetics: Deletion of three of the four alpha-globin genes. Results in excess beta chains forming useless HbH tetramers. Severity: Milder than beta-thalassaemia major. If all four alpha genes are deleted, the result is hydrops fetalis (stillbirth).

Treatment Protocols for Acute Leukaemias

Disorder/Condition	Remission Induction	Consolidation & Maintenance	High-Yield Notes & Protocols
Acute Myeloid Leukaemia (AML)	Intensive Combination Chemo: Standard is Daunorubicin + Cytarabine ("7+3"). Targeted Additions: Gemtuzumab ozogamicin (anti-CD33) for good/standard risk. Midostaurin (FLT3 inhibitor) for FLT3-mutated AML. Older/Frail Patients: Low-dose cytarabine or azacitidine, often with venetoclax (BCL-2 inhibitor).	Consolidation: High-dose cytarabine. Allogeneic HSCT: Considered for poor-prognosis AML (adverse cytogenetics, persistent MRD). Maintenance: Midostaurin (for FLT3+) or azacitidine may be used for some patients not undergoing HSCT.	APML Subtype: Caused by t(15;17). High risk of fatal bleeding (DIC). Treat emergently with All-Trans-Retinoic Acid (ATRA) and Arsenic Trioxide (ATO). Excellent prognosis if initial coagulopathy is managed. Supportive Care: Critical. Manage neutropenic sepsis, bleeding (platelets $>10 \times 10^9/L$), and tumor lysis syndrome (allopurinol/rasburicase). 
Acute Lymphoblastic Leukaemia (ALL)	Combination Chemo: Dexamethasone, vincristine, anthracyclines, asparaginase. Targeted Additions: Dasatinib (TKI) for Philadelphia chromosome-positive (BCR-ABL+) ALL. CNS Prophylaxis: Essential. Intrathecal chemotherapy, high-dose systemic methotrexate.	Consolidation: Blocks of chemotherapy. Allogeneic HSCT: Used for most fit adults with a suitable donor. Maintenance: Outpatient oral/IV chemo for up to 3 years post-consolidation.	Relapsed/Refractory Disease: Novel therapies include Blinatumomab (anti-CD19/CD3 BiTE), Inotuzumab ozogamicin (anti-CD22), and CAR-T cell therapy. Prognosis: Excellent in children (~90% cure). Poorer in adults, especially with high-risk genetics. MRD levels are a powerful prognostic tool.

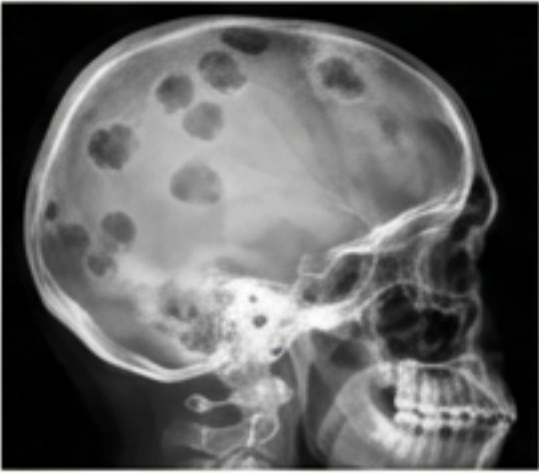
Targeted Therapies for Chronic Leukaemias

Disorder/Condition	1st Line Treatment	2nd Line & Relapse	High-Yield Notes & Protocols
Chronic Myeloid Leukaemia (CML)	Tyrosine Kinase Inhibitors (TKIs): Imatinib, Nilotinib, or Dasatinib. These drugs specifically inhibit the BCR-ABL tyrosine kinase.	Switch TKI: If response is suboptimal or resistance develops (e.g., due to mutations like T315I), switch to a different TKI (Bosutinib, Ponatinib). Allogeneic HSCT: Reserved for patients who fail TKI therapy.	 Hallmark: Philadelphia chromosome t(9;22) creating the BCR-ABL fusion gene. Monitoring: Response is tracked by quantitative PCR for BCR-ABL transcripts. The goal is a major or complete molecular response Phases: Chronic → Accelerated → Blast Crisis (transforms to acute leukaemia). TKIs have dramatically prolonged the chronic phase.
Chronic Lymphocytic Leukaemia (CLL)	Watch and Wait: For asymptomatic, early-stage (Stage A) disease Young/Fit (no TP53 mut.): FCR chemo-immunotherapy (Fludarabine, Cyclophosphamide, Rituximab) Older/Less Fit: Bendamustine or Chlorambucil + Rituximab/Obinutuzumab TP53 mutated: Ibrutinib (BTK inhibitor)	BTK Inhibitors: Ibrutinib, Acalabrutinib PI3K Inhibitor: Idelalisib BCL-2 Inhibitor: Venetoclax (highly effective, risk of tumor lysis) These agents are options for relapsed disease or first-line in TP53 mutated cases	Diagnosis: Clonal B-cell lymphocytosis ($>5 \times 10^9/L$) with CD5, CD19, CD23 expression Prognosis: TP53 mutation or 17p deletion indicates poor prognosis and resistance to chemo-immunotherapy Complications: Hypogammaglobulinaemia (recurrent infections), Autoimmune Haemolysis (Coombs+)

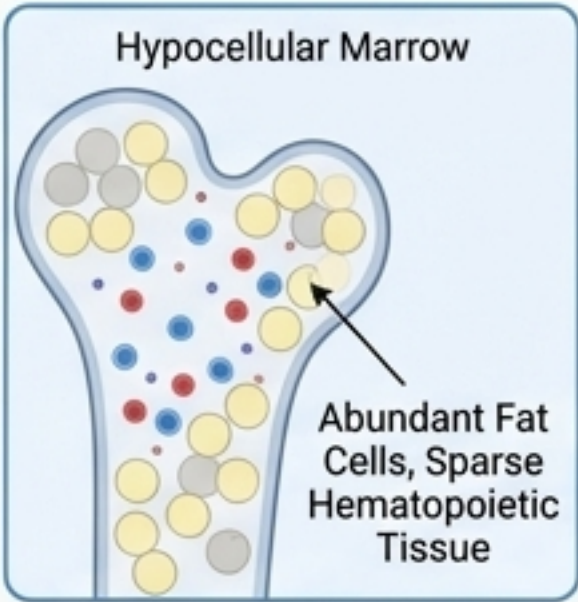
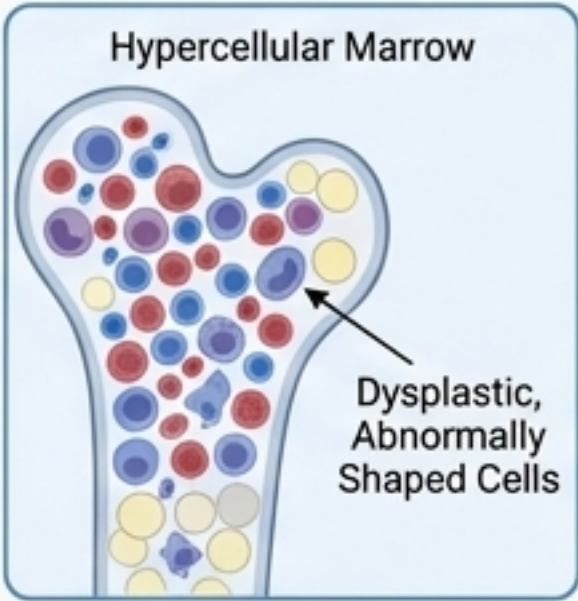
Stage-Adapted Therapy for Lymphomas

Disorder/Condition	1st Line Treatment	Relapsed/Refractory Disease	High-Yield Notes & Protocols
Hodgkin Lymphoma (HL)	Early Stage (IA, IIA): 2-4 cycles of ABVD chemotherapy (Doxorubicin, Bleomycin, Vinblastine, Dacarbazine) ± involved-site radiotherapy. Advanced Stage (IIB, III, IV): 6-8 cycles of ABVD. More intensive BEACOPP regimen may be used. PET-adapted therapy: Interim PET scans guide treatment de-escalation (e.g., dropping Bleomycin) or escalation.	Salvage Chemo followed by Autologous HSCT. Brentuximab vedotin (anti-CD30 antibody-drug conjugate). Checkpoint Inhibitors (Nivolumab, Pembrolizumab). Allogeneic HSCT may be considered.	Hallmark: Reed-Sternberg cells (malignant B-cells) surrounded by reactive inflammatory cells. Staging: Ann Arbor classification is key. "B symptoms" (fever, night sweats, weight loss) indicate more aggressive disease. Goal: Maximize cure while minimizing long-term toxicity (cardiac, pulmonary, second cancers) from chemo/radiation. 
Non-Hodgkin Lymphoma (NHL) - Low Grade (e.g., Follicular)	Watch and Wait: For asymptomatic patients. Symptomatic Disease: Rituximab (anti-CD20) combined with chemotherapy (e.g., R-CHOP, R-CVP, R-Bendamustine). Rituximab Maintenance: Given for 2 years after achieving response to delay progression.	Second-line Chemo-immunotherapy Targeted agents: Idelalisib (PI3K inhibitor). Autologous HSCT: Can produce long remissions in younger patients with early relapse.	Course: Indolent, relapsing-remitting course. Often disseminated at diagnosis and generally not curable with conventional therapy. Can transform to high-grade lymphoma (Richter's transformation).
Non-Hodgkin Lymphoma (NHL) - High Grade (e.g., Diffuse Large B-Cell)	R-CHOP Chemo-immunotherapy: 4-6 cycles of Rituximab, Cyclophosphamide, Doxorubicin, Vincristine, Prednisolone. Radiotherapy: For localized Stage I disease or residual bulk disease after chemo.	Salvage Chemo followed by Autologous HSCT. CAR-T Cell Therapy: Licensed for patients with multiply relapsed disease.	Course: Aggressive, rapidly growing, but potentially curable. Prognosis: The International Prognostic Index (IPI) score (based on age, stage, LDH, performance status) predicts outcome. "Double/Triple Hit" lymphomas with MYC and BCL2/BCL6 rearrangements have a poor prognosis.

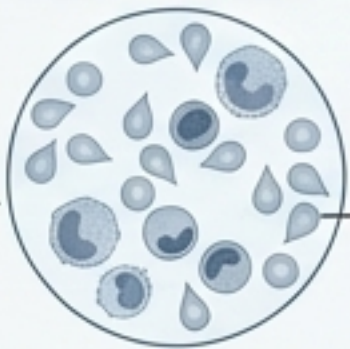
Managing Paraprotein-Related Disorders

Disorder/Condition	1st Line Treatment	Subsequent & Relapse Management	High-Yield Notes & Protocols
Monoclonal Gammopathy of Uncertain Significance (MGUS)	No treatment required. Observation and monitoring.	N/A	Risk of Progression: ~1% per year to myeloma or a related disorder. Monitoring: Patients with an abnormal serum free light chain ratio should be monitored annually for progression.
Multiple Myeloma	Transplant Eligible (Younger/Fit): Induction with novel agents (e.g., Bortezomib/Thalidomide + Dexamethasone), followed by Autologous HSCT. Transplant Ineligible (Older/Frail): Combination therapy, e.g., Melphalan/Prednisolone/Thalidomide (MPT) or Lenalidomide + Dexamethasone. Supportive Care: Bisphosphonates (Zoledronate) to reduce skeletal events, analgesia, hydration.	Lenalidomide Maintenance: Prolongs response after initial therapy. Relapse: Options include Pomalidomide, Carfilzomib, and Daratumumab (anti-CD38 antibody). A second autologous HSCT may be considered.	 CRAB Criteria for Treatment: HyperCalcaemia, Renal insufficiency, Anaemia, Bone lesions. Diagnosis: Requires ≥10% clonal plasma cells in marrow, M-protein in serum/urine, and end-organ damage. Prognosis: Staging (ISS) based on β2-microglobulin and albumin. High-risk cytogenetics (e.g., del17p) are critical.
Waldenström Macroglobulinaemia	Plasmapheresis: For acute hyperviscosity symptoms. Chemo-immunotherapy: Rituximab combined with agents like Bendamustine or Chlorambucil.	BTK Inhibitor: Ibrutinib is highly effective and licensed for use.	Definition: A low-grade lymphoplasmacytic lymphoma producing an IgM paraprotein. Key Features: Hyperviscosity syndrome (nosebleeds, visual disturbance, delirium), anaemia, lymphadenopathy, splenomegaly. Genetics: A high proportion of patients have a mutation in the MYD88 gene.

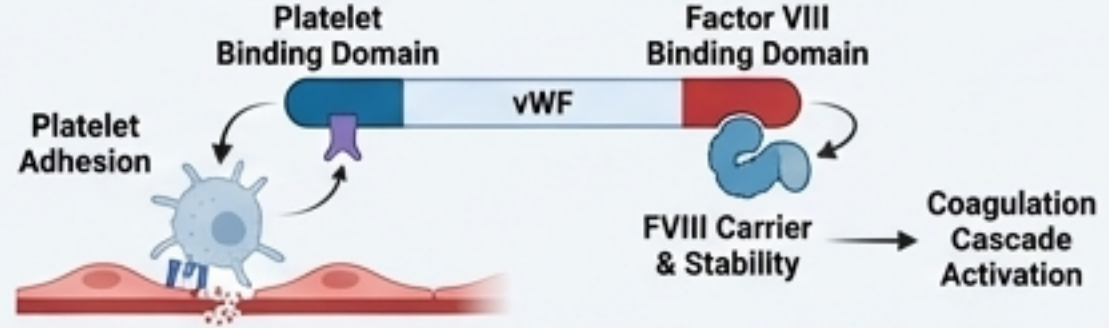
Protocols for Bone Marrow Failure: Aplastic Anaemia & MDS

Disorder/Condition	1 st Line Treatment	2 nd Line & Other Considerations	High-Yield Notes & Protocols	
Aplastic Anaemia	Allogeneic HSCT: Curative treatment of choice for severe disease in patients <35-50 years with a matched sibling donor. Immunosuppressive Therapy (IST): Anti-thymocyte globulin (ATG) + Ciclosporin for older patients or those without a donor.	Unrelated Donor HSCT: For suitable patients who fail IST. Eltrombopag: Thrombopoietin receptor agonist that can induce trilineage responses in patients who fail IST.	Pathophysiology: Autoimmune attack on hematopoietic stem cells leading to pancytopenia and a hypocellular marrow. Severity: Graded by Camitta criteria (neutrophil, platelet, reticulocyte counts). Supportive Care: Blood products and aggressive management of infection are critical. Poor prognosis if only supportive care is given for severe disease.	 Hypocellular Marrow Abundant Fat Cells, Sparse Hematopoietic Tissue
Myelodysplastic Syndromes (MDS)	Low Risk (IPSS-R Low/Intermediate): Supportive care (transfusions), Erythropoiesis-Stimulating Agents (ESAs). High Risk (IPSS-R High/Very High): Azacitidine (hypomethylating agent) improves survival vs supportive care for those ineligible for transplant.	Lenalidomide: Effective for the specific subtype with isolated del(5q). Allogeneic HSCT: The only curative option, considered for younger/fit patients with high-risk disease.	Definition: Clonal hematopoietic disorders with ineffective blood cell production (dysplasia) and risk of progression to AML (~30%). Prognosis: Stratified using the IPSS-R score (based on marrow blasts, karyotype, and cytopenias). Genetics: Associated with clonal hematopoiesis of indeterminate potential (CHIP) in the elderly. Mutations in genes like SF3B1 or TP53 have prognostic significance.	 Hypercellular Marrow Dysplastic, Abnormally Shaped Cells

Managing Myeloproliferative Neoplasms

Disorder/Condition	1st Line Treatment	2nd Line & Other Considerations	High-Yield Notes & Protocols
Polycythaemia Rubra Vera (PRV)	Venesection: To maintain haematocrit <45%. Gives prompt relief of hyperviscosity symptoms. Low-dose Aspirin: Reduces thrombotic risk.	Cytoreduction: Hydroxycarbamide or Interferon-alfa to suppress marrow proliferation, control spleen size, and reduce vascular events. Ruxolitinib: JAK-2 inhibitor licensed for patients not controlled by hydroxycarbamide.	Genetics: >90% of cases have a JAK-2 V617F mutation. Presentation: Symptoms of hyperviscosity (headaches, dizziness), aquagenic pruritus, splenomegaly, or thrombosis (arterial or venous).
Essential Thrombocythaemia (ET)	Low-dose Aspirin: To reduce occlusive vascular events. Cytoreduction: Hydroxycarbamide or Anagrelide for high-risk patients (age >60, platelets >1500x10 ⁹ /L, history of thrombosis).	Interferon-alfa: Has a role in younger patients and during pregnancy.	Genetics: ~50% have JAK-2 V617F mutation, ~25% have CALR mutation. Key Goal: Differentiate from reactive thrombocytosis. Clonal nature, splenomegaly, and specific mutations point to ET. Risk: High risk of thrombosis; can also cause bleeding due to platelet dysfunction.
Myelofibrosis	Supportive Care: Transfusions for anaemia, Folic acid supplementation. Hydroxycarbamide: May help control splenomegaly, high white cell count, or systemic symptoms.	Ruxolitinib: JAK-2 inhibitor effective at reducing systemic symptoms and splenomegaly; may improve life expectancy. Splenectomy: For massive splenomegaly or severe pancytopenia due to hypersplenism. Allogeneic HSCT: Considered for younger patients.	Pathophysiology: Reactive marrow fibrosis driven by growth factors from clonal megakaryocytes. Leads to extramedullary haematopoiesis. Blood Film: Leucoerythroblastic picture with "teardrop" poikilocytes is characteristic.  Leucoerythroblastic Blood Film → Teardrop Poikilocytes

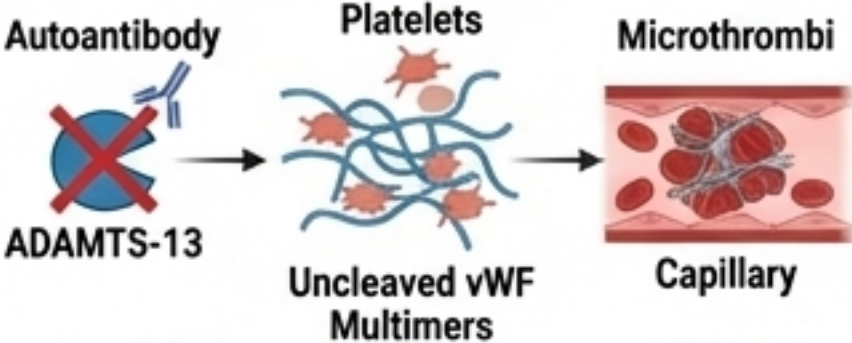
Protocols for Inherited Bleeding Disorders

Disorder/Condition	1st Line Treatment	2nd Line & Other Considerations	High-Yield Notes & Protocols
von Willebrand Disease (vWD)	Desmopressin (DDAVP): For mild/moderate (Type 1) disease. Raises endogenous vWF and FVIII levels 3-4 fold. Tranexamic Acid: Useful for mucosal bleeding.	Factor VIII/vWF Concentrate: For serious bleeds, surgery, or patients unresponsive to DDAVP (e.g., Type 3). Recombinant vWF concentrates are also available.	Most Common Inherited Bleeding Disorder: Characterized by mucocutaneous bleeding (bruising, epistaxis, menorrhagia). Pathophysiology: Quantitative (Type 1/3) or qualitative (Type 2) defect in vWF, which impairs platelet adhesion and FVIII stability. 
Haemophilia A (Factor VIII Deficiency)	Prophylaxis (Severe Disease): Regular infusion of Factor VIII concentrate to prevent spontaneous bleeds. Emicizumab: Subcutaneous bispecific antibody that mimics FVIII activity.	On-Demand Therapy: Infusion of FVIII concentrate at the earliest sign of a bleed. Desmopressin (DDAVP): Useful for mild/moderate disease. Recombinant Activated Factor VIIa (rFVIIa): Used for bleeding in patients who develop FVIII inhibitors.	Inheritance: X-linked recessive. Clinical Feature: Spontaneous bleeding into joints (hemarthrosis) and muscles, leading to chronic arthropathy. Complication: Development of inhibitory anti-FVIII antibodies in ~25% of severe patients, rendering standard treatment ineffective.
Haemophilia B (Factor IX Deficiency)	Prophylaxis (Severe Disease): Regular infusion of Factor IX concentrate. Extended half-life products allow for less frequent dosing (weekly or bi-weekly).	On-Demand Therapy: Infusion of Factor IX concentrate for acute bleeds.	Inheritance: X-linked recessive. Clinical Presentation: Clinically indistinguishable from Haemophilia A. Inhibitors: Less common than in Haemophilia A (<1% of patients).

Management of Venous Thromboembolism (VTE)

Phase / Scenario	1st Line Treatment	2nd Line & Other Considerations	High-Yield Notes & Protocols
Acute VTE (DVT/PE) Treatment	DOACs: Rivaroxaban or Apixaban can be started immediately. Dabigatran or Edoxaban are started after an initial 5-day course of LMWH. OR LMWH followed by Warfarin: LMWH for ≥ 5 days and until INR is therapeutic (target 2.5, range 2-3).	Unfractionated Heparin (UFH): Preferred for massive PE with hemodynamic instability or in patients with severe renal impairment. Thrombolysis: For limb-threatening DVT or massive PE. IVC Filter: For patients with contraindication to anticoagulation or recurrent PE despite therapy.	Cancer-Associated Thrombosis: LMWH is associated with lower recurrence than warfarin. DOACs are increasingly being studied in this population.
Duration of Therapy	Provoked VTE (transient risk factor): 3 months of anticoagulation.  Unprovoked VTE: Extended therapy is often recommended due to high recurrence risk (~30-40% at 5 years). Decision should be individualized based on recurrence risk scores (e.g., DASH) and bleeding risk.	Long-term Anticoagulation: Recommended for patients with ongoing major risk factors like active cancer.	Risk Assessment: All hospitalized patients should be assessed for VTE risk and receive appropriate prophylaxis (mechanical or pharmacological). Post-Thrombotic Syndrome: Occurs in ~30% of patients after proximal DVT, causing chronic leg swelling, pain, and potentially ulceration.

Protocols for Consumptive & Microangiopathic Syndromes

Disorder/Condition	1st Line Treatment	2nd Line & Other Considerations	High-Yield Notes & Protocols
Disseminated Intravascular Coagulation (DIC)	Treat the Underlying Cause: This is the primary and most crucial intervention (e.g., antibiotics for sepsis, delivery for obstetric causes). Supportive Care: Aggressive management of shock, acidosis, hypoxia in an ICU setting.	Blood Component Therapy: Fresh Frozen Plasma, cryoprecipitate, and platelets should be given <i>only</i> if the patient is actively bleeding or requires an invasive procedure. Prophylactic Heparin: Should be given unless clearly contraindicated.	Pathophysiology: Systemic activation of coagulation leads to widespread fibrin deposition (causing organ failure) and consumption of platelets and factors (causing bleeding). Diagnosis: Suspect in the context of sepsis, trauma, malignancy, or obstetric complications. Use ISTH score (platelets, FDPs, PT, fibrinogen).
Thrombotic Thrombocytopenic Purpura (TTP)	Emergency Plasma Exchange: This is a medical emergency. PEX removes autoantibodies and large vWF multimers, and replenishes ADAMTS-13.	Glucocorticoids & Rituximab: To suppress the autoimmune response. Caplacizumab: A monoclonal antibody against the A1 domain of vWF, shown to improve outcomes. 	Pentad: Thrombocytopenia, Microangiopathic Hemolytic Anemia (MAHA), Neurological changes, Fever, Renal impairment. (Few patients have all five). Pathophysiology: Autoimmune deficiency of the ADAMTS-13 enzyme, leading to large vWF multimers that cause microvascular platelet thrombi. Mortality: High mortality (~90%) without treatment.

A Pocket Guide to Hematological Emergencies



Reversing Life-Threatening Anticoagulant-Related Hemorrhage

Warfarin: Prothrombin Complex Concentrate (PCC) + IV Vitamin K.

Unfractionated Heparin: Protamine Sulphate.

Dabigatran: Idarucizumab.

Rivaroxaban/Apixaban: Andexanet Alfa.



Suspected Neutropenic Sepsis in a Chemotherapy Patient

Definition: Fever ($>38^{\circ}\text{C}$) in a patient with neutrophils $<0.5 \times 10^9/\text{L}$.

Action: This is a medical emergency. Do not delay.

Treatment: Administer empirical broad-spectrum IV antibiotics immediately according to local protocols (e.g., piperacillin/tazobactam).



Acute Promyelocytic Leukaemia (APML) Coagulopathy

Recognition: Suspect in any new AML case with severe coagulopathy or DIC.

Action: Do not wait for genetic confirmation.

Treatment: Immediately start All-Trans-Retinoic Acid (ATRA) and provide aggressive coagulation support (cryoprecipitate, platelets, FFP).



Sickle-Cell Crisis: Chest Syndrome or Stroke

Recognition: Acute chest pain, hypoxia, or new focal neurology in a patient with sickle-cell disease.

Action: Urgent intervention required to prevent rapid deterioration or permanent disability.

Treatment: Aggressive supportive care (oxygen, fluids, antibiotics) and urgent Red Cell Transfusion or Exchange Transfusion.